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A Diathesis–Stress Model of Posttraumatic Stress Disorder: Ecological, Biological, and Residual Stress Pathways

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The symptoms captured within the contemporary diagnostic definition of posttraumatic stress disorder (PTSD) have been studied for more than 100 years. Yet, even with increasingly advanced discoveries regarding the etiology of PTSD, a comprehensive and up-to-date etiological model that incorporates both medical and psychological research has not been described and systematically studied. The diathesis–stress model proposed here consolidates existing medical and psychological research data on etiological factors associated with PTSD into 3 causal pathways: residual stress, ecological, and biological. In combination, these pathways illuminate how PTSD might develop and who might be at higher risk for developing the disorder. Research and treatment implications related to the diathesis–stress model are discussed.

Posttraumatic stress disorder (PTSD) is a relatively new name for a historically established set of symptoms. The PTSD symptomatology captured within the most recent edition of the American Psychiatric Association's (1994) *Diagnostic and Statistical Manual of Mental Disorders* (fourth edition; *DSM–IV*) has been described under various labels for more than 100 years. For instance, "railway spine" was diagnosed in survivors of train crashes in the late 19th century, and "combat neurosis" was prevalent among World War I veterans (Shuman, 1995). Such early descriptions of posttraumatic states typically presumed a high degree of blame on the sufferers. Diagnosable individuals were viewed as possessing innate weaknesses that caused them to be more inclined toward pathological responses. In fact, the first edition of the *DSM* (American Psychiatric Association, 1952) described "gross stress reaction," a nomenclatorial predecessor to PTSD, as being a transitory experience unless exacerbated by stable psychopathologies (Elder & Clipp, 1989; McFarlane, 1990). The second edition of the *DSM* (*DSM–II*; American Psychiatric Association, 1968), with its inclusion of "anxiety neurosis," continued the assumption that premorbid vulnerabilities were the primary requirements for diagnosis. It was not until the *DSM*'s third edition (*DSM–III*; American Psychiatric Association, 1980) that the traumatic event itself took diagnostic precedence.

The 1980 revision of the *DSM* led to the recognition that the experience of trauma was the primary etiological factor in a PTSD diagnosis. This dramatic shift in diagnostic criteria clearly reflected a changing social zeitgeist that could be traced back to World War II and that grew more intense during the Vietnam War. Throughout these periods, there was growing academic and social appreciation for individuals directly affected by combat. That is, there was both increasing disapproval of the tempestuous intensity of war and increasing empathy for those who fought (McFarlane, 1990). This zeitgeist seems to have climaxed in the late 1970s and was ultimately reflected in the 1980 revisions to the *DSM* (American Psychiatric Association, 1980).

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The increasingly supportive predilection toward combat veterans not only influenced political and social perspectives, but also the empirical study of PTSD. Researchers had long recognized that the severity of the trauma experience was a primary etiological factor, yet the question of why some people develop a pathological response to trauma while others do not remained relatively unexplored for many years. By focusing on the identification of personal risk factors, it remained possible to overlook the impact of trauma itself, thus leaving trauma survivors to feel totally accountable (to blame) for their reactions. Nonetheless, the severity of the trauma has proven not to be the only etiological factor given that most people do not suffer from prolonged stress reactions in response to events involving threats of severe injury or death. Even among those people who are exposed to the most horrific of traumas such as war (Kulka et al., 1990), violent crimes, traumatic deaths, severe accidents (Norris, 1992), and aggravated assault (Resnick, Kilpatrick, Best, & Kramer, 1992), only a relative minority develop significant PTSD symptomatology. Reported estimates typically range from 5%–35%, and few exceed 50% (McFarlane, 1990).

The possibility of "victim blaming" through an unintentional minimization of the trauma itself arguably affected the direction of early PTSD studies. Researchers began to thoroughly examine and focus on the identification of preexisting risk factors only in the 1990s, and although there is now abundant research on individual risk factors associated with PTSD, only a negligible number of studies have attempted to consolidate different causal pathways into one comprehensive etiological model (see Jones & Barlow, 1990, for an example). Even with existing etiological models established through advanced structural equation modeling (e.g., Fontana & Rosenheck, 1994; Fontana, Schwartz, & Rosenheck, 1997; King, King, Foy, Keane, & Fairbank, 1999), comprehensive integrations of biological and ecological factors, as of yet, remain unrealized. Hence, the purpose of this article is to propose a more inclusive model for understanding the development of PTSD: a diathesis–stress model.

In brief, diathesis–stress models address complex interactions between premorbid risk factors (diatheses) and situational stressors. Diatheses are commonly conceptualized to represent factors that contribute to a constitutional predisposition toward a certain abnormal state or condition. Similarly, situational stressors must be severe enough to activate the diatheses and incite the development of a certain abnormal state or condition, and as the degree or number of diatheses increases, the required severity of a stressor necessary to incite the development of PTSD decreases. Admittedly or not, psychology has been conducting research on diathesis variables of various natures, including ecological and biological ones, since its very beginnings, although the term *diathesis* has not been used as frequently as it is in other scientific fields (i.e., biological, neurological, genetic, and medical research). As noted earlier, there is abundant research on premorbid risk factors specific to PTSD, but very few studies have addressed the effects of a broad range of diatheses in conjunction with situational stressors that are otherwise addressed in diathesis–stress models.

The proposed diathesis–stress model is discussed in more detail in the section that follows. The role of situational stress, ecological diatheses, and biological diatheses are discussed in subsequent sections. Although the material presented in these sections represents only the "tip of the iceberg" in regard to studied and theorized diatheses, it nonetheless opens the door to the possibility that a single model could be used to address the complex interactive effects among ecological diatheses, biological diatheses, and situational stress. Limitations are discussed at the end of each of the diathesis sections. Implications for theory, research, and practice are discussed in the final, and concluding, section.

A Diathesis–Stress Model of PTSD

The relatively universal finding that PTSD develops in a minority of trauma survivors could be attributed to the variability in which certain etiological risk factors are present. Considerable research has identified variables such as premorbid personality characteristics, childhood familial environments, social support, demographics, patterns of psychophysiological stress responses, and severity of trauma (Alarcon, Deering, Glover, Ready, & Eddleman, 1997; Figley, 1978; Kulka et al., 1990). It ap-

pears that the most prominent of these variables could be divided into three etiological pathways—residual (situational) stress, ecological diatheses, and biological diatheses—all of which mutually influence each other.

The stress pathway, originally termed "residual stress" by Figley (1978), reflects the immediate and lingering effects of experiencing a traumatic event. As is generally accepted today, Figley asserted that the severity of the trauma was the primary predictor in the development of PTSD. Therefore, this particular diathesis–stress model proposes that residual stress constitutes the critical catalyst in the development of PTSD. The two remaining pathways, ecological and biological, both reflect diathesis variables, or premorbid risk factors that increase an individual's likelihood of developing PTSD following the experience of trauma and development of residual stress. This model, which diverges somewhat from early diathesis–stress models for other disorders, furthermore proposes that stressors that do not precipitate the development of PTSD are not part of the residual stress pathway. Rather, they have the potential to be incorporated into ecological or biological diatheses. In other words, previous stressors, and their negative psychobiosocial effects, become risk factors in themselves. In such a way, it is the function of any given contributing factor, rather than the chronological placement of the contributing factor, that determines whether it falls into the residual stress pathway or one of the two diathesis pathways.

As part of this model, ecological diatheses encompass risk factors that are linked to an individual's self and surrounding environment (Bronfenbrenner, 1979), such as developmental history, coping mechanisms, modeling and vicarious learning, and interpersonal support systems. Ecological factors can also reflect a host of other environmental and personal variables, including accumulated psychosocial effects of previous trauma experiences (those experiences that do not precipitate the development of PTSD) that have been said to predispose individuals to respond to traumatic events in detrimental ways. Biological diatheses, on the other hand, encompass risk factors such as genetic composition and inherited traits, neurological anomalies, neurochemical and structural alterations (e.g., hippocampal atrophy), and, again, a host of other biophysiological factors, including those associated with chronic or prolonged exposure to stress, that have been said to put individuals at higher risk of developing PTSD following a critical trauma exposure.

Diathesis–stress models of psychopathology assert that all people have some level of predisposing risk factors, or diatheses, for any given mental disorder. However, each individual's "breaking point," or the point at which she or he develops a given disorder, varies depending on the interaction between the degree to which these risk factors are in place and the degree of stress experienced by the individual in question (Monroe & Simons, 1991). In the case of PTSD, it is assumed that the "psychological break" occurs when the trauma is severe enough to activate diatheses. According to the diathesis–stress model of PTSD being proposed here, individuals who are most at risk for developing PTSD would have greater degrees of diathesis variables (ecological, biological, or both). Thus, individuals with higher degrees of premorbid risk factors (diatheses) would not need to experience as severe a stressor to reach the breaking point and develop PTSD symptomatology. In contrast, individuals with extremely low degrees of diatheses might not display any signs or symptoms of PTSD after experiencing a traumatic event. This model could consequently be used to explain why some people develop PTSD and others do not.

The proposed diathesis–stress model accordingly asserts that ecological and biological diatheses interact with each other and with the residual stress pathway and, in such a way, constitute complex interaction effects in the development of PTSD (see Figure 1). Although both ecological and biological pathways serve as diatheses, or premorbid risk factors, the residual stress pathway remains as the necessary catalyst for the potential onset of PTSD.

The nature of the interactions between an individual's degree of diatheses and the effects of residual stress is extremely complicated. Research examining residual stress pathways suggests that as the intensity of the trauma (stressor) increases, so too do the risks for pathological responding. This implies a discrete linear dose–response relationship (Snow, Stellman, Stellman, & Sommer, 1988). However, Monroe and Simons (1991) asserted that the relationship between variables is not that simple. They noted that diathesis–stress models represent additive

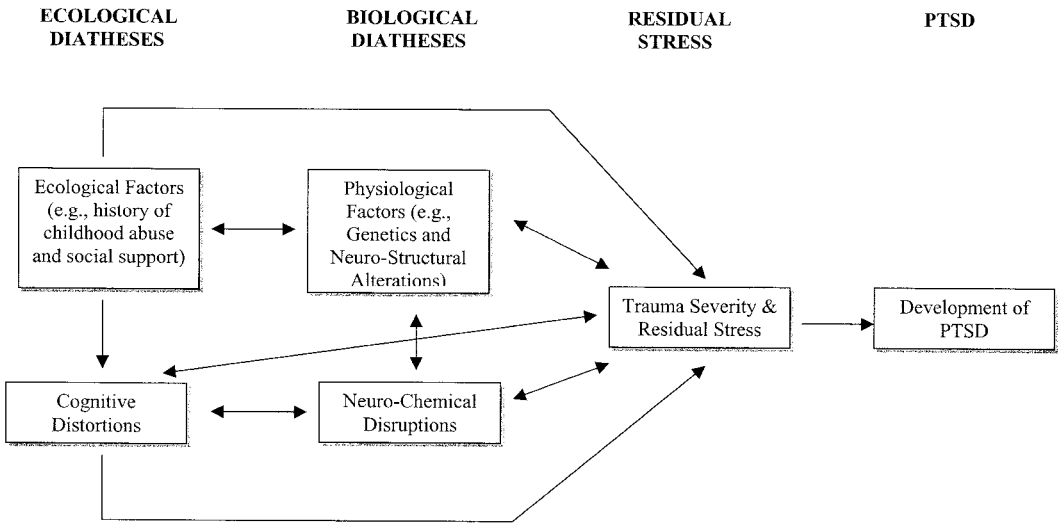

Figure 1. Proposed diathesis–stress model for posttraumatic stress disorder (PTSD). Developmental, biological, and residual stress factors are included.

and interactional relationships between variables. This complexity is partly due to the complicated nature of, and linked relationships between, diatheses. The diatheses involved in PTSD, as well as other psychological disturbances, tend to be both multifarious and somewhat ambiguous. In addition, the list of potential diatheses can appear infinite upon initial consideration, thereby highlighting the importance of continuing studies designed to clarify the exact factors, many of which are highlighted in this article, that are reliably associated with PTSD.

Despite the problems in identifying and measuring diathesis variables, contemporary psychologists, including traumatologists, are beginning to capitalize on the value of diathesis–stress models. Research to date has yet to examine the efficacy of this model with PTSD, although several studies have examined the possibilities of using such a model to understand the development of various other psychological problems and disorders. For example, several researchers have examined the efficacy of using a diathesis–stress model to understand the development of depression (Banks & Kerns, 1996; Coyne & Wiffen, 1995; Metalsky & Joiner, 1992; Monroe & Simons, 1991; Spangler, Simons, Monroe, & Thase, 1993, 1996, 1997). In addition, researchers have explored the efficacy of this model in relation to schizophrenia (Walker & Diforio, 1997), maladaptive achievement and helplessness (Boggiano, 1998), and suicide (Dixon, Heppner, & Anderson, 1991; Schotte, Cools, & Payvar, 1990). From a logical point of view, it makes sense that such a model might also be valuable in understanding disorders, such as PTSD, that are ultimately and commonly believed to have been brought on as a result of experiencing extreme stress.

Role of Residual Stress

Foy, Carroll, and Donahoe (1987) described residual stress as being a common negative psychological condition resulting from the experience of a traumatic event. Severity of outcome is said to be the direct result of degree of trauma experienced. Accordingly, greater levels of residual stress are the result of a more severe trauma experience. This ideology is reflected in the current diagnostic criteria that must be met for a diagnosis of PTSD. According to the *DSM–IV* (American Psychiatric Association, 1994): "The person [must have] experienced, witnessed, or [been] confronted with an event or events that involved actual or threatened death or serious injury, or threat to the physical integrity of self or others . . . [and] the person's response involved intense fear, helplessness, or

horror" (pp. 427–428). The assumption is that a person must, first and foremost, experience an extremely intense trauma to develop PTSD.

On the basis of a comprehensive review of literature investigating etiological factors related to PTSD in Vietnam combat veterans, Foy et al. (1987) concluded that "the best etiological predictor of PTSD found in the research literature appears to be degree of combat exposure" (p. 25). King et al. (1999) provided compelling support for this conclusion through structural equation modeling. Using data from both male and female Vietnam veterans, their results revealed direct links between PTSD and pretrauma (e.g., family instability and childhood antisocial behavior), trauma (e.g., war-zone stressors), and posttrauma (e.g., social support) variables, as well as direct links between pretrauma and posttrauma variables. Moreover, their findings suggested that war-zone stressors (severity of combat trauma) were predominant in influencing the development of PTSD among male veterans, whereas posttrauma resiliency factors such as lack of social support appeared to be the most significant causal pathway for female veterans. These results are quite meaningful in that they support the contention that trauma severity is a critical factor in the development of PTSD while also supporting the current proposition that other factors, such as social support, might be equally important in understanding the development of PTSD.

Fontana and Rosenheck (e.g., 1994) have produced similar statistical support for the residual stress model using structural equation modeling. Their analyses revealed four latent factors associated with the development of PTSD among Vietnam veterans: (a) premilitary (e.g., family instability or history of abuse), (b) military (combat related versus non–combat related), (c) homecoming and reception (e.g., rejecting welcome by society or low support from family and friends), and (d) postmilitary traumas. The resulting data suggested that the two most significantly and directly related "causal" factors were exposure to combat and familial support after discharge from the military. Societal rejection upon homecoming was also an influential factor. Thus, like King et al. (1999), Fontana and Rosenheck found that the trauma itself was of great importance, but not the sole factor in the development of PTSD.

Evidence of the complex interaction between residual stress and other variables has not only come from research with combat veterans, but also from research concerning survivors of rape and childhood sexual abuse. There is considerable indication that more severe violations (i.e., intercourse vs. unwanted touching) are associated with higher ratings of personal traumatization in victims of abuse (Browne & Finkelhor, 1986). The psychological reverberations of childhood sexual abuse can also be different depending not only on the severity of the abuse but also the nature of the familial response to the abuse. Browne and Finkelhor (1986), in a thorough literature review, found that negative parental reactions tend to intensify the child's trauma. High levels of social support for female rape victims likewise seem to diminish the psychological fallout, as well as decrease the psychological recovery time (Steketee & Foa, 1987). In sum, research strongly suggests that the residual stress pathway interacts with other diatheses, such as pretrauma and posttrauma environment, in the development of PTSD.

Ecological Diatheses

Research clearly suggests that personal, family, cultural, and social environment plays an important role in the development of PTSD. Accordingly, distressing ecological factors ranging from interpersonal style to mainstream discrimination practices, group marginalization, limited social support, and private family chaos could potentially act as viable ecological diatheses. Whereas diathesis–stress models for other disorders might consider stressful environmental variables, such as group marginalization, to fall in the residual stress category, this model clearly reserves the residual stress category for the stressor or trauma that causes the "psychological break" (i.e., ultimately leads to the development and symptom expression of PTSD). All other stressful variables are therefore considered to be incorporated into an individual's ecological adjustment or biological development. Thus, the history of a stressor will automatically become a diathesis. Similarly, the deleterious biological impact of stressors becomes a biological diathesis, and the deleterious psychosocial impact of stressors can become an ecological diathesis.

For example, a history of childhood physical or sexual abuse has been identified as a substantial factor in the development of PTSD later in life and, therefore, a potential ecological diathesis (e.g., Kulka et al., 1990; Zaidi & Foy, 1994). Zaidi and Foy (1994) similarly found a significant, positive correlation between childhood physical abuse and combat-related PTSD in veterans. Duncan, Saunders, Kilpatrick, Hanson, and Resnick (1996), in a random sample of American women, also found that women with histories of childhood physical abuse were nearly 5 times more likely to have a lifetime history of PTSD, and 10 times more likely to currently be experiencing PTSD, than women without histories of physical abuse. In fact, Bremner, Southwick, Johnson, Yehuda, and Charney (1993) found that Vietnam veterans with combat-related PTSD and Vietnam veterans without PTSD differed on only 2 of 12 precombat variables examined in their study: physical and sexual abuse in childhood. Engel et al. (1993) conducted a similar study with front-line male and female Gulf War veterans. Their results revealed that whereas male veterans with abuse histories had slightly higher rates of combat-related PTSD, female veterans with abuse histories had significantly higher rates of combat-related PTSD than female veterans without such histories. Comparable studies have shown that nonmilitary women who experienced childhood sexual abuse are likewise more inclined to suffer from PTSD later in life (e.g., Saunders, Villeponteaux, Lipovsky, Kilpatrick, & Veronen, 1992).

Engel et al. (1993) reported that exposure to familial abuse sometimes results in the development of maladaptive coping skills, feelings of helplessness, vulnerability, and loneliness. These characteristics are consistent with combat-related PTSD (Z. Solomon, 1993). Moreover, these characteristics have been linked to increased risk for future victimization (Boney-McCoy & Finkelhor, 1995, 1996). Interestingly, Zaidi and Foy (1994) suggested "that punishment history and combat exposure may not be wholly independent factors" (p. 38). That is, the positive correlation between severity of physical abuse in childhood and PTSD symptomatology later in life might be complicated by a higher risk for placement in potentially traumatic situations. For instance, Green, Grace, Lindy, Gleser, and Leonard (1990) reported that premorbid psychopathology was predictive of happenstance involvement in more extreme combat-related situations. This finding is further complicated by the suggestion that parental abuse can be predictive of later antisocial behaviors and decreased social skills (McCord, 1983; Pollack et al., 1990), which have in turn been linked to higher risk for more severe trauma exposure (e.g., Elder & Clipp, 1989; Zaidi & Foy, 1994). It is therefore possible that a history of abuse predisposes an individual not only to placement in more traumatic situations, but also to the maladaptive cognitive patterns theoretically associated with the development of PTSD (McFarlane, 1990).

Maladaptive cognitive styles and perceptual distortions are accordingly linked to histories of childhood abuse. Moreover, maladaptive cognitive styles, perceptual distortions, and histories of childhood abuse represent ecological diatheses for later onset of PTSD (note that these diatheses are likely interdependent). A history of childhood abuse has in fact been linked with maladaptive social information processing. For instance, Dodge, Pettit, Bates, and Valente (1995) found that abused children develop unique cognitive patterns such as a relative unawareness of nonhostile social cues, a hypervigilance to hostile social cues, and a greater tendency to attribute hostile intent to other children's behaviors.

Similar social processing biases have been linked to the later development of PTSD in the face of trauma. One maladaptive cognitive pattern that has, in particular, been widely associated with the development of PTSD is learned helplessness. Prolonged and perceivably uncontrollable trauma tends to incite overly helpless, if not self-defeating, behaviors in humans (Fincham & Cain, 1986; Peterson, Maier, & Seligman, 1993), and quite distressingly, childhood abuse is one such circumstance that is often prolonged and uncontrollable in the child's eyes. Thus, children who experience abuse tend to develop helpless thought patterns that are eventually generalized to other similar situations (Bolstad & Zinbarg, 1997).

It has been suggested that cognitive distortions developed in childhood as a result of abuse often persist into adulthood (Jehu, 1992) and, as suggested by the proposed diathesis–stress model, negatively influence the experience of trauma later in life. Bolstad and Zinbarg's

(1997) study on female survivors of childhood sexual abuse and sexual assault linked learned helplessness with the onset of PTSD. Their results suggested that a history of childhood sexual abuse was significantly associated with a decreased sense of control over external events. This decreased locus of control, conceptually similar to learned helplessness, was also associated with increased severity of PTSD symptoms following sexual assault experiences in adulthood. Regehr, Cadell, and Jansen (1999) similarly found that long-term recovery in women who survived a rape or an attempted rape was partially predicted by global perceptions of locus of control. Moreover, global perceptions of uncontrollability served as stronger predictors of problematic recovery than ratings specific to the rape, whereas greater belief in personal controllability was associated with lower levels of psychological distress. In fact, Frye and Stockton (1982) established that an external locus of control accounts for 12% of the variance in PTSD symptoms among Vietnam veterans.

Unfortunately, it is not always clear exactly when, and as a result of what trauma, PTSD develops. This comes from the fact that diatheses appear to be latent, particularly ecological diatheses. However, Monroe and Simons (1991) warned that diatheses are rarely entirely latent. As such, individuals with histories of childhood abuse who also experience trauma later in life might consequently have been exhibiting unnoticed symptoms consistent with PTSD before the full development of PTSD following the adult trauma. It becomes difficult to determine precisely what role—correlational, causational, or otherwise—childhood history plays in the development of adult-onset PTSD for any given individual. In addition, not all individuals who are exposed to childhood abuse develop symptoms of PTSD or become revictimized later in life; thus these suppositions do not hold true for all cases. It is possible, then, that the experience of childhood stressors can have no significant effect or could be incorporated into a resiliency, thereby precluding its inclusion in a diathesis pathway. In cases in which revictimization does occur, it is unclear as to whether this is due to an inherent predisposition (i.e., personality or traits), some form of learned stimulus response (i.e., learned helplessness), maladaptive cognitions that lead individuals to interpret situations differently, or other uncontrollable and totally external factors such as group marginalization. There is also debate over the extent to which cognitive patterns, personality traits, and behaviors are influenced by environmental or genetic factors. For example, is learned helplessness actually learned, or are some people genetically predisposed to emotionally concede sooner than others? This debate is further complicated by the possibility that psychophysiological vulnerabilities and resiliencies can be influenced by a combination of biological, ecological, and situational (e.g., exposure to trauma or prolonged stress) factors.

Biological Diatheses

Developing certain cognitive and behavioral response patterns, such as dissociation or hyperarousal, can be highly adaptive in response to acute traumas. However, prolonged exposure to traumatic stimuli or consistently reinforced behaviors, including social information-processing biases, could result in permanent changes in neural networking (Perry, Pollard, Blakley, Baker, & Vigilante, 1995). Children are especially susceptible to such changes given that there are critical periods in the development of the human brain marked by increased neural plasticity in childhood (Gazzaniga, Ivry, & Mangun, 1998). In such a way, "the more a child is in a state of hyperarousal or dissociation, the more likely they are to have neuropsychiatric symptoms following trauma" (Perry et al., 1995, p. 271).

Recent advances in technology have enabled researchers to examine biological factors thought to be associated with prolonged exposure to trauma, such as child abuse, as well as the development of PTSD. The results of these examinations have resulted in exciting, albeit controversial and arguably enigmatic findings on how stress can critically alter brain volume, neuronal architecture, hormonal makeup, and the like. For instance, early research by Mason, Giller, Kosten, and Harkness (1988) examined neuronal activity in adults diagnosed with PTSD. They found that male PTSD participants, as compared with participants diagnosed with other psychiatric disorders (e.g., major depressive disorder and schizophrenia), evidenced higher 24-hr urinary excretions of both norepi-

nephrine and epinephrine. In addition, cortisol levels were lower, and norepinephrine–cortisol ratios were more elevated. These findings were noteworthy given that abnormalities in norepinephrine and serotonin levels have been specifically linked to the increased anger, hostility, and depression associated with PTSD. Increased norepinephrine levels in the locus coeruleus have also been associated with the activation of traumatic memories (Southwick, Krystal, Johnson, & Charney, 1995).

Other research has similarly supported the assertion that catecholamines and their respective receptors play a critical role in the expression of affective and anxiety disorders. For instance, research findings involving military and nonmilitary trauma victims suggested a strong possibility "that cyclic adenosine monophosphate (AMP) signal transduction may be reduced [in individuals with] PTSD" (Lerer, Bleich, Solomon, Shalev, & Ebstein, 1990, p. 150). This supposition of Lerer and his colleagues was supported by a second study conducted with Lebanon War veterans diagnosed with PTSD and nonclinical controls (Lerer et al., 1990). Results revealed that the PTSD participants had significantly reduced Forskolin-stimulated adenylate cyclase activity in platelet membranes, which strongly suggested "the possibility that PTSD could be associated with an intrinsic dysfunction in cyclic AMP signal transduction and the level of the catalytic unit of the receptor-adenylate cyclase complex" (p. 154). Of note, increased catecholamines have been associated with high levels of psychomotor agitation that, when combined with the significantly high levels of stress-related hormones and neurotransmitters associated with PTSD, might increase an individual's gross susceptibility to pathological responses in the face of trauma.

Putnam and Trickett were able to directly link history of abuse with psychobiological tendencies in a 1997 study that included data on sexually abused girls, nonabused controls, and girls who had experienced general maltreatment (*M* age = 11 years, *SD* = 3). They specifically established the presence of significant levels of hypothalamic–pituitary–adrenal axis (HPA) dysregulation and decreased hippocampal volume in the abused girls. Heim, Owens, Plotsky, and Nemeroff (1997) explained similar findings by reporting that "it is plausible that alterations in stress response neurobiological systems might be the link between stressful experiences and the development of psychopathology in the genetically vulnerable individual" (p. 195). It is furthermore possible that such vulnerability could be linked to a history of childhood abuse.

The study of HPA dysregulation and PTSD is a remarkably dynamic and growing field because of the possible link between HPA functioning and an individual's increased biological susceptibility to the disorder. That is, the HPA is partially regulated by the corticotrophin-releasing factor (CRF), which in turn is responsible, to some degree, for the regulation of stress reactions (Heim et al., 1997). Heim et al. noted that CRF produces psychophysiological effects such as increased heart rate, increased locomotor activity, potentiated acoustic startle response, enduring sensitization to psychosocial stimulants, and enhanced fear conditioning. Related findings reviewed by Heim et al. suggested that CRF is responsible not only for the regulation of stress reactions, but more specifically for the regulation of arousal, vigilance, and behavioral inhibition. These behaviors are correspondingly key diagnostic aspects of PTSD. As such, hyperactive CRF levels have been found in several traumatized populations, including Vietnam veterans with PTSD (e.g., Bremner, Licinio, et al., 1997).

Chronic exposure to traumatic stress has furthermore been associated with structural changes in specific areas of the brain, including the hippocampus (e.g., Gurvits et al., 1996), although much debate continues to surround clinical interpretation of associated findings (Bremner, 2001; McEwen, 2001; Yehuda, 2001). Human studies in this area have been driven by animal research, which shows that prolonged exposure to stress-induced glucocorticoids alters hippocampal neuronal morphology (Wooley, Gould, & McEwen, 1990). Using such animal research as a springboard, Bremner and colleagues (1995) found greater degrees of hippocampal atrophy among Vietnam veterans diagnosed with PTSD than among matched controls with the help of modern brain scanning technology, specifically magnetic resonance imaging. Bremner, Randall, et al. (1997) replicated the pattern with adult survivors of childhood physical abuse and similarly found significantly reduced hippocampal volume. They posited that the reduced hippocampal volume

evident in both veterans and abuse survivors could be, among other possibilities, a result of prolonged exposure to extreme stress. Stein, Koverola, Hanna, Torchia, and McClarty (1997) also conducted a study with female survivors of childhood sexual abuse and further substantiated the occurrence of reduced hippocampal volume in survivors of prolonged stress. Sapolsky (1996) explained these results, noting that prolonged exposure to stress-induced glucocorticoids seems to result in atrophy to subcortical regions of the brain. In such a way, the very same hormones that excite and save us at times of extreme danger can eventually, with prolonged exposure, degenerate the area of the brain (hippocampus) responsible for translating short-term memories into long-term storage.

Although the evidence for biological pathways to PTSD is quite compelling, it is not yet clear whether these pathways are causal or reactive factors. As multiple researchers, most notably Sapolsky (1996), have pointed out, research has yet to conclusively determine which comes first, PTSD or neurobiological anomalies. Moreover, the exact mechanisms of neurological change, including contradictory findings on lower versus higher cortisol levels in acutely and chronically stressed humans, continue to elude modern research (Yehuda, 2001). Likewise, psychiatric diagnoses comorbid with PTSD, such as substance dependency, may complicate our understanding of biological diatheses (Bremner, 2001).

One other factor that is often ignored in existing biological literature and complicates our understanding of biological diatheses involves sex differences. Although it is commonly accepted that "sex" is a biological factor or a phenotype (with visible physical characteristics) and that "gender" is more of a genotype (reflecting the totality of influential factors, including biological, but most notably ecological influences), it is often difficult to separate their respective effects on biological functioning. The terms are often applied interchangeably, thus making it difficult to determine what is actually being measured in some neurobiological research. It is also nearly impossible to measure the effects of one without including the influence of the other, especially in instances in which diathesis–stress interactions are being studied. The relative effects of sex versus gender differences may therefore actually constitute separate diathesis pathways.

Summary and Implications

Even in response to the most devastating traumas, only a relative minority of individuals develop prolonged psychological disturbances such as PTSD. Evidence clearly dictates that those who do develop such persevering disruptions in psychological well-being are different from those who do not on a multitude of factors. That is, the nature and severity of the trauma is clearly the primary causal factor, but not the sole etiological pathway to the development of PTSD. Research enumerates several additional etiological pathways, or groups of diatheses, that interact with the precipitating traumatic experience. The diathesis–stress model proposed here organizes these additional diatheses into two separate, yet related pathways—ecological and biological—that have been shown to increase the likelihood that an individual will develop PTSD following the experience of trauma. An individual will therefore have an increasingly higher likelihood of experiencing residual stress, or the lingering adverse effects of trauma exposure, as the number of that individual's preexisting ecological or biological diatheses (i.e., risk factors) increases. Such lower thresholds for residual stress, by the very definition of residual stress, will furthermore result in reliably higher rates of PTSD development.

This theory of PTSD development has some novel features relative to early diathesis–stress models for other mental disorders, and although such a novel approach can be controversial in its inherent limitations (as outlined in previous sections), it can also serve to improve our understanding of how PTSD develops in some people but not others. The novel nature of this model is most apparent in the suggestion that the experience of a stressor does not necessarily qualify as *the* stressor in the diathesis–stress model. In other words, this model suggests that a stressor can incite the development of PTSD, thereby falling into the residual stress pathway, or the history of the stressor and its negative psychobiosocial effects, if not severe enough to actually incite residual stress and the associated development of PTSD, can be incorporated into a person's ecological adjustment or biological development, thereby falling into the ecological

or biological etiology pathway, or both. This model further suggests that a history of a significant stressor, such as childhood abuse, will automatically fall into a diathesis pathway, but psychobiosocial effects of that same stressor, such as cognitive biases or neurochemical changes, do not necessarily have to qualify as diatheses. Rather, this model implies that any given stressor has the potential to have no significant and lasting psychobiosocial effect. Likewise, any given stressor might actually encourage positive psychobiosocial effects, thereby precluding any inclusion of these effects in either of the two diathesis (i.e., risk factor) pathways. Such neutral or resiliency factors are not highlighted in the proposed model because it presumes that the degree of diatheses and their combined interactions with residual stress remain the developmental key to PTSD.

The consolidation of three etiological pathways—residual stress, ecological, and biological—is similarly unconventional in that it calls for a multidisciplinary approach to understanding PTSD. Psychological research has traditionally been divided across detached research specialties (i.e., clinical psychology, medical psychiatry, developmental studies, etc.). The proposed diathesis–stress model, however, incorporates neoteric findings from each of these specialties and asserts that a combined approach is necessary to gain an accurate understanding of the development of PTSD. This multidisciplinary conceptualization is timely, given the more recent call for cooperation between and among disciplines. Moreover, a multidisciplinary research approach would likely be much more successful at addressing outcome anomalies and theoretical weaknesses than a nonunified research approach that only examines a singular etiological pathway. The implications of this approach are especially pertinent to the increased use of structural equation modeling in the identification of "causal" pathways.

Within the area of PTSD research, several decidedly landmark structural equation models have focused on developmental or residual stress variables without yet addressing the possible interaction with biological variables (e.g., Fontana & Rosenheck, 1994; Fontana et al., 1997; King et al., 1999). Future empirical examinations of this diathesis–stress model, optimally through the use of additional structural equation modeling, would therefore deliberately include measures of both biological and ecological diatheses. In addition, future examinations can serve to clarify how the ecological and biological pathways combine with one another and residual stress to ultimately incite the development of PTSD. As suggested in previous sections of this article, a clarified formula for how ecological and biological diatheses, as well as residual stress, interact with each other continues to elude modern researchers, although an integrated developmental model can potentially serve as a grounding force in the examination of integrated statistical analyses of diatheses.

By considering the concurrent influences of residual stress, ecological diatheses, and biological diatheses on the development of PTSD, applied practice should in turn be affected. Unfortunately, clinical practice (e.g., diagnostic assessments, preventive treatment, or tertiary treatment) has been criticized for focusing on either the biological–anatomical side of a disorder or the ecological–psychosocial side of a disorder without consistent integration of the two, despite compelling support for such an approach (S. D. Solomon, Gerrity, & Muff, 1992). Psychiatrists, psychologists, and social workers alike have also been cited for underutilizing empirically based treatments in clinical practice despite compelling research findings informed by clinically relevant theoretical models of disorder development (e.g., Sanderson, 2002). Therefore, it would be important to not only carefully test the clinical applicability of integrated interventions but also provide clinicians with readily accessible information on the anticipated benefits of integrated approaches. Wide dissemination of this information might support mental health treatment programs that are working toward creating or improving efficacious multidisciplinary approaches to the assessment and treatment of PTSD.

This diathesis–stress model for PTSD similarly has the potential to help illuminate who should be targeted for preventative interventions, what risk factors should be assessed, and the different treatment modalities that might best address the respective risk factors. Because the model highlights the importance of premorbid risk factors, markedly high-risk individuals might be more readily identifiable based on the degree to which these risk factors are in place. Moreover, although the model does not necessarily enable practitioners to reach survivors of

traumas any quicker, it could be valuable in helping practitioners to identify appropriate (and therefore more effective) interventions after an individual has been identified. For instance, a practitioner (of one of many disciplines) might be able to separate high-risk individuals and low-risk individuals following exposure to a manmade or natural disaster into an intensive follow-up group (i.e., psychiatric consultations, psychological treatment, social work assessments, and primary care referrals) versus a less intensive follow-up group (i.e., brief supportive counseling). This identification process would be in contrast to many existing treatment practices that are reactive to the development of PTSD, as opposed to proactive to the potential (and costly) development of the disorder.

Similarly, this identification process contrasts with the traditional focus on one causal pathway at a time (e.g., pharmacological therapy without treatment addressing environmental factors, and vice versa). In fact, typical psychological and pharmacological interventions, when used alone, are often not comprehensive enough to adequately treat individuals with PTSD (S. D. Solomon et al., 1992), which can result in higher personal and health care costs in the long run. This model accordingly asserts that practitioners across all disciplines who have contact with trauma survivors must not only consider the immediate effects associated with residual stress, but also consider past abuses and neglect, familial relationships, support systems, cognitive patterns, potential biological anomalies, and other diathesis factors.

Finally, a diathesis–stress formulation of the development of PTSD is beneficial because it inherently places an emphasis on the primary etiological role of residual stress, above and beyond the individualized risk factors encompassed in diathesis pathways. This emphasis is not only an accurate reflection of empirical evidence on the etiology of PTSD but also a reminder that the disorder, at its core, is a result of an external trauma. In such a way, the intrinsic design of this diathesis–stress model acknowledges the exceptionally turbulent and often uncontrollable nature of traumas that typically incite the development of PTSD. Ideally, then, this model could work to minimize the possibility of erroneous "victim blaming" while propelling theory-driven research that will continue to improve our understanding of what puts people at higher risk for developing this devastating disorder.

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Correction to Rudmin (2003)

The article "Critical History of the Acculturation Psychology of Assimilation, Separation, Integration, and Marginalization," by Floyd W. Rudmin (*Review of General Psychology*, 2003, Vol. 7, No. 1, pp. 3–37), contained several errors.

On page 11, left column, line 22, *Lock* should be *Loeb.* On page 15, Table 3, the acculturation label *colonialism* for No. 50, Berry et al. (1972), and all of the acculturation labels for No. 51, Zak (1973), are Rudmin's wording, as the original scholars did not label those categories of acculturation. On page 16, right column, 2nd paragraph, lines 9–13, eight scholars are identified as psychologists who did not well cite prior research on acculturation. In fact, Hoffman was a philosophy doctoral student and Campisi was a sociology doctoral student. On p. 18, left column, first paragraph, line 5, there is a statement that "earlier versions of this $2 \times 2 \times 2$ taxonomy were not cited." Berry (2001, p. 619) did cite the 1974 paper, but not in reference to defining the new taxonomy. On page 32, the Devereux and Lock (1943) reference should instead be to Devereux and Loeb. On page 35, the correct pagination for the Taft (1953) reference is 45–55.

This critical history began by noting faults in the psychometrics and research results of contemporary acculturation research and then engaged a history of acculturation in order to gain insight into the origins of those faults. The work of John Berry, as the most prolific contemporary scholar of acculturation psychology and as the leading advocate of the fourfold paradigm, was salient in these criticisms. Readers should note that Berry's (2003) most recent explanation of the fourfold theory gives good coverage of many of the concerns raised in this critical history. For example, the origins of the acculturation constructs prior to his entry and during his own career are discussed. A focus on minority rights is articulated, as well as the need to consider economic success as part of the concept of adaptation. The influence of political ideology on his own research and that of others is presented. See Berry, J.W. (2003). Conceptual approaches to acculturation. In K. M. Chun, P. B. Organista, & G. Marín (Eds.), *Acculturation: Advances in Theory, Measurement and Applied Research* (pp. 17–37). Washington, DC: American Psychological Association.